Developing an Evidence-Based Model for Concussed Military Personnel with PTSD: The Application of Psychophysiological Assessment and EEG/QEEG/ERP

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This TeleSeminar on Comorbid PTSD/TBI

- Presents research data explaining treatment challenges
- Proposes a personalized treatment model that
- Uses quantifiable physiological and electrophysiological data
- In order to individually time the progression of treatment
- For the purpose of
  - Developing an evidence-based treatment model that
  - Restores pre-combat level functioning
  - In a minimal period of time while
  - Reducing costs and
  - Reducing veteran homelessness and suicide rates
What is Not Going to be Discussed

- Debate that TBI & PTSD can coexist
- Definitions of TBI & PTSD
- The majority of military that do not experience long-term problems with TBI
- I will not delineate what is common known about PTSD and TBI
Topics to Cover

- “Invisibly Wounded Warrior” phenomenon
- A New Theoretical Basis
- PTSD Progression in the Brain
- TBI Progression in the Brain
- Accumulative Issues of Coexisting PTSD/TBI
- Shortcomings in Scientific Literature
- Identified Areas of Research Needs
- Comorbid Treatment Considerations
- Psychophysiological Assessment Application
- EEG/QEEG/ERP Application
Topics to Cover

- Timing Treatment Based on Physiological and Electrophysiological Data
- Phase I: Returning ANS to Normal
  - Biofeedback Relaxation Therapy
- Phase II: Returning Cognitive Functioning to Normal
  - QEEG-Guided Neurotherapy
- Summary
Caring for Invisibly Wounded

- “In country”
  - No visible wounds, no treatment
  - Many experience multiple blasts
  - Minimal recovery time

- “Invisibly Wounded Warriors” are often:
  - Treated like malingerers or outcasts
  - Denied Purple Hearts
  - Over medicated
  - Disciplined for inability to pull duty
“Invisibly Wounded Warrior” (IWW) Veterans

- Many are discharged and many are unable to work
- Some cannot even drive
- Many have to rely on parents for financial support
- IWWs are referred to VA for services
- VA backlog delays resolution of disability claims
- Texas VA has a backlog of 35,000 MEBs
- VA admits system is overloaded with IWWs
- VA is shorthanded in mental health staff
TBI/PTSD

- PTSD strongly associated with mild TBI
  - New England Journal of Medicine (Hoge, January 2008)

- Dr. Bart Billings predicts that the PTSD/TBI wounds will cost the U.S. two hundred billion dollars per year in care
  - http://mcia-inc.org/resources/TBI-PTSD+$28GI+WILSON$29.ppt
Theoretical Underpinnings

- E. Roy John’s EEG Homeostatic Regulatory System
- The deviating from homeostasis
  - Creates pathology
  - Predetermined by an identifiable EEG/QEEG pattern (EEG Phenotype)
- Prolonged deviation results in a new homeostatic set point
  - The brain adapts to a war/survival set point
  - Which may take years to reset without help
EEG Phenotypes
(Gunkelman, 2008)

Prior studies using EEG have documented “clusters” of EEG/qEEG features within psychiatric populations


Phenotypic Patterns are *Not* Isomorphic with the DSM Categories

Phenotypes have powerful implications for both medication, and neurotherapy


Phenotypes Have Led to Enhanced Outcomes Clinically

- S. Suffin and H. Emory (attentional and affective, medication)
- L. Prichep (OCD, medication)
- Chabot, et al. (ADD/ADHD, medication)
- C. Wright (ADD/ADHD, Neurofeedback)
- M.I.N.D. (U.C. Davis, Autism)

Databases for EEG are starting to include the genetic component (Brain Resource Company, Australia)
Development of PTSD

- Most individuals are able to cope with traumatic exposure and do not develop PTSD.

- Neurological soft signs (NSS) have been associated with increased risk of developing PTSD.
Neurological Soft Signs and PTSD
Gervitz et al.

- (2000) PTSD subjects correlated NSS with more
  - neurodevelopmental problems
  - childhood ADHD systems
  - lower IQs
- (2006) concluded that
  - neurologic dysfunction in PTSD does not reflect brain damage acquired along with the PTSD
  - but instead represents a familial vulnerability factor, which likely antedates the traumatic exposure
Neuroanatomical Differences May Account for NSS

- Reduced hippocampal volume
- Increased amygdala reactivity and decreased connectivity with the PFC
- Reduced volume of the frontal cortex
- Large cavum septum pellucidum
NSS + EEG Phenotype + Trauma = PTSD

• NSS are neurodevelopmental structural anomalies

• Three EEG phenotypes are correlated with CNS overarousal

• Trauma exposure
  • Dysfunctions the limbic system (sympathetic dominant)
  • Excitatory neurochemistry predominates

• The trauma exposure triggers the onset of PTSD in those that are predisposed to CNS overarousal
EEG Phenotypes in PTSD Associated with CNS Hyperarousal

- **Beta Spindles:** COMP-0 genetic correlate

- **Very fast alpha peak frequency:** Alpha frequency $>11$ Hz, commonly 12, 13 and even at 14 Hz

- **Low Voltage Fast (LVF):** common in alcoholics and seen with gene #4 Gaba receptor changes
Neurochemical Differences in PTSD

- Decreases in cortisol concentration (adrenal fatigue)
- Increased concentration of corticotropin-releasing factor (CRF)
- Blunted adrenocorticotropic hormone (ACTH) response to CRF stimulation
  - NE hyperactivity (increased startle response)
  - Increased DA (fear conditioning)
Neurochemistry Changes in PTSD

- Increased glutamate
  - inhibits retrieval of emotional memories
  - may promote consolidation of traumatic memories
- Reduced cortisol promotes CRF-NE cascade
  - and enhances prolonged stress response
- Lack of GABA, 5-HT and NPY
  - accentuate stress responsiveness
- Increase NE enhances
  - encoding of fear memories
Electrophysiology of Blast Exposure

- Damages connections between white and gray matter (cortex and subcortex)
- Cortical damage slows peak frequency of alpha dramatically (6 to 7 Hz)
  - Which is a thalamic-cortical issue
- Gray matter injury
  - Reduces output of alpha, beta, gamma
  - Alpha returns in 6 to 12 months
    - But is usually slowed
  - Beta and gamma remain in deficit
Neuroanatomical Impact of Blast TBI

- Often involve the orbital frontal surface (subgenual) and to
- The lower temporal area which
  - Disrupts normal SNS-PNS balance to sympathetic dominance due to damage to subgenual area which
  - Opens the thalamic gate which
  - Exaggerates the sensory ERP components due to lack of thalamic gating
Traumatic Brain Injury Progression

- Phase I: Initial mechanical damage
- Phase II: Progressive deterioration of the neural axis
- Phase III: Recovery
Initial Mechanical Damage Phase I

- Rupture of cellular and vascular membranes
- Release of intracellular contents
- Cessation of blood flow
- Cascade of cellular events compromising injured and uninjured brain regions
Progressive Deterioration Phase II

- The events include:
  - Release of excitatory amino acid neurotransmitters (*glutamate* & *aspartate*)
  - Activation of glutamate receptors
  - Influx of calcium (+CA ions)
  - Free-radical generation
  - Inflammation
  - Downstream events lead to cell injury and death (apoptosis)
Neuronal Apoptosis

• Neuronal apoptosis may be an adaptive response to conserve resources needed for healing.

• Continued physical challenge and cognitive challenge increases demands on resources for healing.

• Hypothesis: If cognitive and physical challenge increases neuronal apoptosis, then cognitive and physical rest should decrease recovery time compared to those who continue physically and cognitively challenging activities.
Neurochemical Differences in TBI

- Decreased concentration of 5-HT (impulsivity, hostility, depression, and suicidality)

- Increased glutamate release (overexposure can result in excitotoxicity)

- Decreased plasma Neuropeptide Y (maladaptive stress response)

- Increased activation of the endogenous opioid system (numbing and dissociation)
TBI & Cognitive Functioning

- The diffused axonal injury in TBI:
  - Slows processing speed
  - Inhibits memory function
  - Disorganizes network communication and thus
  - Disrupts cognitive functioning
- Diminished cognitive abilities renders most PTSD treatment ineffective
- May cause hyperarousal, increasing suicidal risks
Recovery Phase III

- Phase 1: involves reversal of inhibition of function and initiation of cell repair
- Phase 2: entails change in the properties of existing pathway
- Phase 3: involves the formation of new connections. (Note: Treatment strategies that enhance neuronal activity may facilitate cognitive recovery)
- Apoptosis continues for at least 22 weeks
- Symptoms can be delayed for 18 months
TBI Evidence-Based Treatment

- Cognitive and physical rest
- Prevention of further head trauma
- Management of existing symptoms and
- Education as to healing process and potential for pathology
Strategies Supporting Recovery

- Early pathway activation of
- Noradrenergic (relaxation),
- Dopaminergic (reward) and
- Cholinergic (lipid enriched diet) and
- Cortical stimulation and
- Physical exercise (also promote plasticity and growth factors)
Cumulative Effects of Comorbid TBI & PTSD

**TBI**
- ANS becomes sympathetic dominant
- Increases toxic levels of glutamate
- Decrease of Neuropeptide Y
- Decrease of 5-HT concentration
- Reduces cognitive abilities

**PTSD**
- ANS becomes sympathetic dominant
- Increases toxic levels of glutamate
- Decrease of Neuropeptide Y
- Decrease of 5-HT concentration
- Reduces cognitive abilities
Evidenced Based Treatment and Co-Morbidity

- Currently “no empirically validated therapies exist to treat co-morbid PTSD, depression, and post concussive disorders, which may be confounded by self-medicated alcohol misuse, abuse, or dependence.”.

Identified Shortcomings in Scientific Literature When PTSD and TBI Coexist in Military

- No published studies on accuracy of diagnostic tests
- No studies on evaluation of efficacy or harm of treatment
- No studies on psychopharmacological intervention
- The quality of identified studies is generally fair however, external validity is generally poor.
- Insufficient evidence required to make high quality diagnostic and treatment recommendations does not exist
Identified Areas of Need in Treating Military with Comorbid PBSD & TBI

- Objective measures that limit ascertainment, recall or reporting bias
- Outcome measures of clinically-relevant biological, psychological and social indicators based on trauma etiology, sub-types and time from trauma
- Adequately powered, high-quality randomized controlled treatment trails to evaluate clinical effectiveness and harm of potential therapeutic options
Comorbid PTSD/TBI Treatment Considerations

- TBI and PTSD cause the limbic system to become dysfunctional
  - Disregulated HPA axis appears to be a long-term predictor of suicide (Jokinen et al. 2009)
- TBIs diminish cognitive abilities
  - Render PTSD treatment ineffective
- TBIs inhibits effectiveness of psychiatric medication for treatment of PTSD
Timing is Everything

- Each individual is different
- There is no one size fits all
- Treatment must be designed for each individual
Psychophysiological Assessment to Track Autonomic Nervous System Function

- PPA measures biological functions of:
  - Heart rate
  - Respiration rate
  - Peripheral blood volume
  - Galvanic skin response
  - Muscle tension
- And compares baselines to startle response, math challenge and trauma re-exposure
- Correlates with CNS functioning
Quantitative EEG (QEEG) for TBI

• QEEGs are sensitive (but not specific) to grey and whiter matter injury

• QEEGs identify changes but cannot identify if recent brain insult caused the changes

• Evoked Related Potentials (ERP) are more appropriate for differential diagnosis
  • ERPs can rule out TBI based on ICA analysis of the ERP
Evoked Related Potentials (ERP) are more appropriate for differential diagnosis. ERPs can rule out TBI based on ICA analysis of the ERP.
Evidence-Based Treatment Model for Comorbid PTSD/TBI

- **Phase I:**
  - Rebalance the ANS
  - Restore sleep
  - Family reintegration
  - Teach compensatory strategies
  - Start exercise program

- **Phase II:**
  - Reregulate neurological functioning
  - Stimulate neurogenesis
  - Restore cognitive functioning
  - Resolve traumatic issues
Phase I: Returning ANS to Normal Functioning

- Cognitive and physical rest
- Reregulate the Autonomic Nervous System
- Relaxation training (biofeedback)
- Start QEEG-Guided Neurotherapy
- Psychoeducation
- Skills group training
- Family reintegration therapy
- Aerobic exercise (without exacerbating TBI SXs)
Biofeedback Relaxation Therapy

- Biofeedback can enhance synchronous activity and will facilitate neurointegration.
- Heart Rate Variability biofeedback is an efficient and effective (measurable) tool to return the limbic system to homeostasis.
- However, the person must be in a place of safety and know that he will remain safe until his brain heals and rewires.
When to Employ PTSD Evidence-Based Treatment

- Early cognitive intervention may increase suicidal risks
- Cognitive based therapies that could elicit a stress response and should not be employed until the ANS is re-regulated and cognitive abilities have returned
- Once the SNS-PNS balance is reestablished (identified by their PPA), Phase 2 can proceed
- Pharmacotherapy can be used to manage symptoms however, caution must be taken in regard to side-effects (more prevalent in TBI)
Phase II: Restoring Cognitive Functioning to Normal

- Continue with neurogenesis stimulation
  - Voluntary physical exercise
  - Increase cognitive challenge (novelty)
  - Prosocial activities (great life experiences)
- Continue QEEG-Guided Neurotherapy
- Proceed With Process Group Therapy
- Trauma Focused Cognitive Therapy
- Couples Therapy (if applicability)
Quantitative EEG & QEEG-Guided Neurotherapy

- Can identify the disconnected networks and abnormal power distribution in the brain.
- This information can provide brain computer interface (BCI) training protocols to encourage adaptive plasticity restoring normal brain function.
- Post-QEEGs can be use to measure restoration of normal functioning.
- Cognitive psychometrics can confirm restoration.
Needed Research

- Need to identify a non-invasive method of monitor apoptosis progression

- What is a good quantifiable measure of cognitive functioning and what is the minimal level needed to employ cognitive based therapies?

- How can we measure if a brain is fully healed from a TBI in order to avoid a secondary-impact injury from returning to duty too soon?
Summary

• Loss of connectivity and diffused neuron loss (apoptosis) from TBI results in loss of cognitive processes which inhibits PTSD treatment
• PTSD exacerbates TBI symptoms
• TBI must be treated first
• Each brain injury is different and healing progression is specific to the individual
• Personalizing medicine for the military with comorbid PTSD and TBI is likely to
  • Optimize treatment,
  • Improve prognosis and ultimately
  • Reduce costs
Surviving War is Tough

Surviving the Memories is Tougher on Those Left Behind